Short Communication

Contrast Echocardiography in the Diagnosis of Myocardial Stunning

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Summary: The identification of viable myocardium within dysfunctional myocardium has important clinical implications. By using a microvascular tracer, myocardial contrast echocardiography may have the potential for prediction of myocardial viability in the acute and subacute phases of myocardial infarction. In the case presented, the normal myocardial perfusion observed after intravenous injection of the contrast agent, combined with severe wall motion abnormality following prolonged chest pain, suggested myocardial stunning. This was confirmed by normal coronary angiography and by restoration of normal left ventricular function at 1-month follow-up echocardiography.

Key words: contrast echocardiography, myocardial perfusion, myocardial ischemia, viable myocardium

Introduction

The identification of viable myocardium in the setting of acute myocardial infarction (MI) or chronic coronary artery disease with reduced left ventricular function has important prognostic and therapeutic implications. Many noninvasive techniques have been studied for this purpose, such as single-photon-emission computer tomography perfusion imaging

with 201-thallium or 99m-technetium-labeled perfusion agents, positron emission tomographic imaging of perfusion and glucose uptake, low-dose dobutamine echocardiography, and, recently, contrast echocardiography for evaluation of microvascular integrity. New second-generation contrast agents and new techniques such as harmonic and intermittent imaging for the detection of these agents have been developed, creating the possibility of clinical introduction of contrast echocardiography.^{1,2}

Case Report

In March 1998, a 75-year-old woman was sent to CNR, Institute of Clinical Physiology, Pisa, for chest pain lasting for 2 h with the suspicion of an acute MI. She had had no chest pain previously but had been treated previously for elevated blood pressure. On physical examination at admission she had pallor, rhythmic pulse of 84/min, blood pressure of 130/70 mmHg, systolic murmur of mitral regurgitation without pulmonic rales. The electrocardiogram (ECG) showed 2 mm ST-segment elevation in leads V₂–V₄ without R-wave reduction or pathologic Q waves. Echocardiography performed on admission revealed a left ventricular aneurysm including the distal half of the left ventricle, mild mitral and aortic regurgitation, and a calculated ejection fraction of 35% (Fig. 1A). After informed consent was given, contrast echocardiographic study, as part of an ongoing international investigation, was performed in the catheterization laboratory just before angiography. Echocardiographic contrast agent NC100100 (Nycomed Amersham, Oslo, Norway) was injected intravenously at a dosage of 0.20 ml, using the right cubital vein, followed by 10 ml of saline flush. The pictures captured in intermittent harmonic mode by commercially available Sonos 2500 (Hewlett-Packard Co., Andover, Mass.) echocardiographic equipment were stored on magneto-optical disc. The left ventricular aneurysm was seen on the left ventricular phase of contrast echocardiography, using apical four-chamber and two-chamber views. After a few beats, the contrast agent appeared in the left ventricular myocardium. A complete opacification of the left ventricular myocardium, including the walls of the aneurysm, could be observed without any perfusion defect (Fig. 2). The

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FIG. 1 (A) Two-dimensional (2-D) echocardiography performed before coronary angiography showing the left ventricular aneurysm in the apical two-chamber view. LV= left ventricle. (B) Follow-up 2-D echocardiography: Normal left ventricular function, no wall motion abnormality.

patient was referred for primary percutaneous transluminal coronary angioplasty with the suspicion of left anterior descending coronary artery occlusion based on history and ECG findings. Left ventriculography confirmed the aneurysm of the left ventricle; however, left and right coronary angiograms revealed normal coronary arteries without any angiographically important lesions (Fig. 3).

The patient was treated medically. Her cardiac enzymes showed mild elevation (CPK: 292U/l, CPK-MB: 46U/l, SGOT: 84U/l, SGPT: 64U/l, LDH: 490U/l). Her ECG at discharge as well as at repeat echocardiography were unchanged.

At 1-month follow-up, the patient had no symptoms of chest pain; the ECG showed negative T waves in anterior leads, and control echocardiography showed normal left ventricular function (ejection fraction 70%) without any wall motion abnormality (Fig. 1B).

Discussion

In patients post acute MI, left ventricular dysfunction may indicate necrotic myocardium, viable tissue, or a combination of both. Myocardial ischemia may be reversed with coronary artery reperfusion; however, the viable myocardium may demonstrate prolonged but transient contractile dysfunction.³ By using a microvascular tracer, myocardial contrast echocardiography may have the potential for prediction of myocardial viability in the acute and subacute phases of MI.⁴ The microvascular integrity, detected by myocardial contrast echocardiography (MCE) as contrast enhancement in dysfunctional segments, is the basis of functional recovery at follow-up. Kloner *et al.*⁵ noted that with MI, myocyte loss is accompanied by a loss of microvasculature. Accordingly, the absence of myocardial opacification with MCE may be evidence of a

FIG. 2 Myocardial contrast echocardiography before coronary angiography showing no perfusion defect.

FIG. 3 Coronary angiography with normal left coronary arteries.

lack of myocardial viability. The recovery of contractile function in the reperfused area after MI takes place within the first few days after reperfusion and depends on the status of myocardial perfusion in the dysfunctional segments.

Myocardial stunning represents a condition in which coronary blood flow is restored after an ischemic episode, but myocardial functional, biochemical, and ultrastructural abnormalities persist for a period. In our case, the normal myocardial perfusion detected by contrast echocardiography combined with severe wall motion abnormality following prolonged chest pain suggested myocardial stunning. Negative coronary angiography correlated with the result of contrast echocardiography and left ventriculography confirmed the left ventricular aneurysm detected by echocardiography. Myocardial stunning is a common situation in clinical practice, but there are no data available for early recovery of left ventricular aneurysm after MI. Left ventricular aneurysm can disappear after MI by spontaneous recanalization or angioplasty. A small series of three cases was reported in which the left ventricular aneurysm disappeared late after acute MI (5 months–3 years).⁶ This recovery is compatible with hibernation, not stunning. Aneurysm formation with normal coronary arteries is uncommon. Among consecutive 1,800 postinfarction patients, a Japanese group found 5 patients with left ventricular aneurysm and no or minimal coronary arterial obstruction;⁷ however, there are no follow-up data on the later change of the aneurysm.

The intracoronary use of MCE was reported earlier for the study of the extent of residual perfusion within the infarct area. At 6-month follow-up echocardiographic study, an improvement in regional and global function was observed only when residual perfusion, detected by MCE, was present in the infarct zone.⁸

In our case, the echocardiograms performed at admission and 7 days later at discharge revealed no change in left ventricular wall motion. The restoration of contractile function took place between 1 week after the ischemic period and the 1-month follow-up. The ST elevation during chest pain persisting for few days and the mild elevation of cardiac enzymes suggested some subendocardial necrosis, which may have contributed to the delay of wall motion normalization. The

transient negative T waves persisting for weeks after ischemia were reported to be consistent with “subendocardial stunning,” and they can be documented frequently in patients after prolonged ischemia.⁹

The unique feature of our case is stunning confirmed by myocardial contrast echocardiography resulting in left ventricular aneurysm formation and the complete restoration of left ventricular function observed at follow-up echocardiography.

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